TABLE II

ROUTINE WASSERMANN REPORTS

Hospital Obstetrics

Year	No. of Diagnosis	Negative	Positive	Per Cent Positive
1919	289	280	9	3.1
1920	297	287	10	3.3
1921	243	232	11	4.5
1922	217	208	9	4.1
1923—6 mo.	107	103	4	3.7
Total....	1153	1110	43	3.7

TABLE III

STERILITY

	Primary Clinic	Primary Private	Secondary Clinic	Secondary Private	Total	Per Cent
Number ...	83	53	26	38	200	
Infected ...	61	27	20	24	132	.66
Cured	9	2	0	7	18	.09
INFECTIONS						
Bartholinitis	5	9	1	6	21	.10
Skenitis ...	8	10	4	6	28	.14
Cervicitis ..	50	23	15	17	105	.52
Pelvic Inflammatory	30	13	9	8	60	.30
Appendicitis	19	13	3	8	43	.21
OTHER CONDITIONS						
Retroversions	26	12	11	14	63	.31
Fibroids ...	14	6	4	3	27	.13

TABLE IV

TESTS OF PATIENT AND HUSBAND FOR CAUSES OF STERILITY

	Primary Clinic Pt.	Primary Clinic Husb.	Primary Private Pt.	Primary Private Husb.	Secondary Clinic Pt.	Secondary Clinic Husb.	Secondary Private Pt.	Secondary Private Husb.	Totals Pt.	Totals Husb.
Reubin Test.....	0		5		0		4		9	
Tubes Closed.....			3				1		4	
Wassermann Test.	47		0		16		4	2	67	2
" Positive	8	1*			3	1*	0	0	11	2
Condom Test.....		5		10		6		12		33
Aspermia		3		5		5		3		16
Gonorrhoea		7*		1*		3*		1*		12

*No test made. Diagnosis based upon history only.

THE SIGNIFICANCE OF CERVICAL PATHOLOGY IN STERILITY*

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The material for this report has been collected from patients seen at Stanford women's clinic and from the private records of Professor Spalding. No attempt has been made to select particular types of cases and these records have been compiled just as the cases presented themselves, in order to obtain some idea of the relative frequency of the various lesions contributing to the production of sterility in the female. Only cervical conditions presented by the patients studied are considered.

In 230 sterile women examined, whose average age was twenty-nine years, and whose average period of sterility was five years, chronic infection of the cervix was present in 129, or 56 per cent, and of these 129 cases, seventy-two or 31 per cent, gave palpable evidence of internal pelvic inflammation. Undoubtedly, many more than this had internal pelvic inflammatory disease, for it is often not palpable. In thirty cases of chronic endocervicitis we found closed tubes by insufflation tests in twenty-two, or 73 per cent of cases. (Table I.)

Stenosis of the cervix appears as the possible cause of the sterility in eleven cases. The importance of cervical stenosis in the production of sterility is questionable, and alone is probably rarely a cause. In each of the eleven cases here reported, there were varying degrees of inflammation, apparently enough in each case to produce sufficient thick mucus to plug the stenotic canal.

The seven infantile cervices (Table I) accompanied infantilism of the other organs of generation and probably should not be listed as a separate cause of sterility. An endocervical polyp was the cause of sterility in one case. A glandular hyperplasia of the endometrium was also present. This patient became pregnant shortly after curettage of the uterus and cervix.

Forty-four of these cases were tested for tubal patency. Of fourteen tested who showed no signs of cervical infection, all tubes were found to be patent; in thirty infected cervices, only eight showed patent tubes, while twenty-two admitted no gas whatever at several testings. The eight inflammatory cases with patent tubes probably owed their sterility to the cervical condition directly. The possibility of endometrial or ovarian inflammation is not excluded, however. (Table II.)

In a series of 125 cases, average age 26, with known cervical inflammation, forty-two were sterile. In each one of these cases a section of the cervix and a curettage specimen of endometrium were studied microscopically; in only sixteen was there any evidence of infection, thus showing the marked immunity of the endometrium even in the presence of cervical and tubal disease. (Table III.)

Infection of the intact cervix is practically always gonorrhoeal at its inception, often later being entirely replaced by other organisms; lacerations and contusions of the cervix which do not heal promptly predispose the cervical tissues to infection by other pyogenic organisms.

The presence of chronic infection in the glandular elements of the cervix changes the mucus secreted by the cervical glands into thick mucopurulent plugs which entangle the spermatozoa and also kills them. The reaction of the secretion is often changed from mildly alkaline to strongly acid, possibly contributing somewhat in reducing the vitality of the spermatozoa.

Local plugging of the cervical canal, however, is not the most important role played by cervical infections in producing sterility. The cervix is the distributing focus for practically all infections of the internal organs of generation. Lymphatic

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drainage from the mucous surface of the cervical canal is upward and outward through the musculature of the uterus toward the serous coat, from where the lymphatic vessels leave the uterine surface by way of the sacro-uterine and broad ligaments, and it is this path by which the infection reaches the pelvic organs. The milder infections of the cervical glands seem to cause the production of thicker mucus plugs, while the more virulent infections cause a more serious discharge but extend to the adnexae earlier and with more disastrous results.

In these 230 sterile patients, pelvic inflammation seems to be the most important single condition causing sterility, showing 56 per cent infected. The indication is for early and thorough treatment of the cervix to prevent local barriers to the ingress of spermatozoa and to prevent internal extension of the infection which may cause permanent destruction of the generative organs.

TABLE I

STERILITY—230 CASES

Av. Duration Sterility	Average Age	Cervicitis	Pelvic Inflammatory Disease	Stenosis	Infantile	Polyp
5 yrs	29	129	72	11	7	1
		56%	31.3%	4.7%	3.0%	0.4%

TABLE II

TUBAL PATENCY RESULTS

44 Sterile Cases

Cervix	Number	Tubes Open	Tubes Closed
Clean	14	14	0
Infected	30	8	22

TABLE III

CERVICITIS—125 CASES

Average Age 26

Cervicitis	Pelvic Inflammatory Disease	Endometritis	Primary Sterility
125	46	16	42
100%	37%	13%	33.6%

THE ADNEXAL ORGANS IN RELATION TO STERILITY*

By C. W. ANDERSON, M. D., Los Angeles

The increased knowledge of the physiology and pathology of the ovary, which has come with the wonderful advances made in the study of the endocrine system, as well as the epoch-making contribution of Rubin to the diagnosis of tubal conditions, have given an unprecedented impetus to the study of the whole subject of sterility.

The tubes and ovaries play a very important part in the production of sterility and until comparatively recently the blame for almost all cases of sterility was laid at the door of the tubes.

I would like to discuss this subject under the headings:

1. Pathological conditions of the tubes and ovaries causing sterility.
2. Their diagnosis.
3. Their treatment.

It is hardly necessary for me to say, by way of introduction that one of the conditions necessary for impregnation is a tube offering an open channel for the ovum to descend and the spermatozoon to ascend, and that any condition which interferes with these functions is a potential cause of sterility.

Infantilism occasionally manifests itself in a persistence of the spirally twisted condition of the tube, which is common in early life. This may cause sufficient obstruction in the lumen of the tube to prevent the free passage of the ovum or spermatozoon and hence be a cause of sterility. In most cases of this kind evidences of infantilism are present in other parts of the generative system, and make it all the more improbable that pregnancy can occur.

Acute appendicitis in childhood or early womanhood may be a factor in producing sterility in later life. Graves (Archives of Surgery 1921-2, 315) has particularly called attention to this subject. He believes that very frequently the serofibrinous exudate from the region of the inflamed appendix gravitates to the pelvis and sets up sufficient peritonitis to cause occlusion of the abdominal ostium of the tube. This would explain many of our cases of occluded tubes in which no history of gonorrhoeal or other pelvic infection is obtainable.

Salpingitis is by far the most important factor in impairing the functional value of the tube as an oviduct. Curtis (Surgery, Gynecology and Obstetrics, December, 1921) gives the results of the bacteriological and pathological investigation of fallopian tubes removed at operation from 300 patients. Not only were smears made from the fluid in these tubes, but the tubes themselves were ground up and cultured. He concluded that the gonococcus was responsible for approximately 80 per cent of these cases, other pus-producing bacteria, notably various types of streptococci, caused 15 per cent, while evidence of tuberculosis was found in 5 per cent.

In this discussion we are especially interested in the end results of infections, for it is these which cause sterility. It was formerly believed that when once a patient had developed gonnorrhoeal salpingitis she was doomed to permanent closure of both tubes with resulting sterility. More careful study of these cases has proven that gonorrhoeal salpingitis, more than any other type of salpingitis, shows a tendency to spontaneous recovery, and that many women with bilateral gonorrhoeal salpingitis have recovered and subsequently borne children. A single attack of mild gonorrhoeal salpingitis frequently leaves no bad after effects. Those tubes, however, which are subjected to a severe attack of salpingitis, or frequent reinfection from a focus, such as an endocervicitis, are apt to be permanently damaged. In such cases the fimbriated extremity very frequently becomes occluded either by retraction or adhesion of the fimbriae, or by a plastering of the end of the tube to the ovary, or some adjacent structure.

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